Intradural Aneurysm Caused by Arterial Injury during Surgery

Treatment with Coil Embolization

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Summary

We described a patient with subarachnoid hemorrhage due to a ruptured intradural aneurysm caused by arterial injury during surgery and severe symptomatic vasospasm. The iatrogenic posttraumatic aneurysm was occluded by using GDC with preservation of parent artery followed by intraarterial infusion of papaverine to treat vasospasm as an one-stage procedure.

This resulted in significant angiographic resolution of the vasospasm and the patient's clinical outcome was excellent. Endovascular approach to posttraumatic intracranial aneurysms and endosaccular GDC embolization of aneurysm with concomitant intraarterial papaverine infusion to treat vasospasm are discussed.

Introduction

Traumatic intracranial aneurysms frequently occur after head injury, but they also can be found after iatrogenic arterial iniury during surgical procedures. These aneurysms are usually difficult to eliminate without sacrificing the parent artery either surgically or by endovascular route [1,2]. Recent advances, however, allow safe approaches to complicated intracranial vascular lesions, such as Guglielmi detachable coil (GDC) embolization of extra- or intradural posttraumatic aneurysms with preservation of the parent artery [3-5].

Case Report

A 50-year-old woman was evaluated preoperatively with angiography because of the right sphenoid wing menengioma encircling the right distal carotid artery (figure 1A). Angiography with ipsilateral carotid compression showed good blood flow through the circle of Willis without additional vascular abnormality (figure 1B). The patient underwent successful operation without any complication. Computed tomography (CT) obtained on the same day showed no hemorrhage either in the craniotomy site or other intracranial spaces. Although postoperative clinical course was uneventful, the patient returned to the hospital two weeks later with clinical and CT findings of subarachnoid hemorrhage (SAH) (figure 1C). She was in Hunt and Hess, grade III., Angiography showed 5-mm ipsilateral carotid artery saccular aneurysm at the origin of the ophthalmic artery without marked vasospasm. The patient, however, gradually deteriorated within 24 hours and developed mild left hemiparesis 48 hours later. The decision was made to treat the aneurysm via the endovascular approach and try to treat the vasospasm during the same procedure. In the third day after admission, angiography showed severe spasm of the M1 segment and distal internal carotid artery on the affected side (figure 1D). With the patient under general anesthesia and heparinization, we advanced a microcatheter and a 0.010 inches

guide wire coaxially in a 6F guiding catheter positioned in the right internal carotid artery. Embolization of the aneurysm was successfully performed with 17-cm GDC-10 microcoils. Embolization was followed by infusion of total dose of 150 mg papaverine over 20 minutes into the middle cerebral artery and distal internal carotid artery just proximal to the narrowed segments via the same microcatheter. This resulted in significant angiographic resolution of the vasospams (figure 1E). The patient's clinical status improved slightly, and she still had a mild left hemiparesis on discharge. After rehabilitation the hemiparesis resolved nearly totally. An angiogram 3 months later confirmed complete occlusion of the aneurysm.

Discussion

Posttraumatic intracranial aneurysms arise from a wide range of conditions, including penetrating or blunt head trauma, transsphenoidal surgery for tumor or sinus disease, and unintentional laceration of the arteries after craniotomy for any reason. Many authors tend to call all such traumatic aneurysms as pseudoaneurysm. Posttraumatic aneurysm, indeed, is divided histologically into two types: true aneurysms and pseudoaneurysms [6]. True aneurysms arise from partial damage to the arterial wall with an intact adventitia. Pseudoaneurysms are more common and develop when the entire arterial wall is lacerated and surrounded by a hematoma. It is organized and encapsulated by inflammatory cell proliferation in subacute and chronic phase, that is it has pseudowall. In our case, the arterial laceration was not seen during surgery. Additionally, neither immediate postoperative CT scan, nor CT obtained after SAH showed intracranial hematoma, instead, the latter showed only SAH. We, therefore, define our patient as having iatrogenic (posttraumatic) intradural true aneurysm. The development of iatrogenic aneurysm after surgery, even in the absence of visible arterial injury is sometimes unavoidable, since arterial wall injury may somehow occur during surgery in case of the carotid artery entrapped by the tumor [7]. The differentiation of these two types of traumatic aneurysm is important not only to give them correct name but also to decide the timing of therapy. Lempert et al have emphasized the importance of age of pseudoaneurysm in determining the relative safety of endosaccular aneurysm occlusion, since subacute pseudoaneurysms have pseudowall that are more mature and strong [3].

Many posttraumatic intracranial aneurysms are pseudoaneurysm and extradural in origin. Cavernous or petrous segment aneurysms are usually presented with cranial nerve palsies, carotico-cavernous fistula, and lifethreatening epistaxis. They may occur in head trauma with cranial base fracture or as a complication of transsphenoidal surgery. Intradural aneurysms are presented with SAH and associated with closed head trauma or iatrogenic arterial injury during surgical procedures. They are either pseudoaneurysm associated with hematoma or true aneurysm when the causative injury weaken the arterial wall only. The natural history of psaudoaneurysms is diverse. They may expand gradually, rupture in a few weeks or months, or spontaneously shrink and disappear [1,6]. In reviewing the literature, Dario et al found that 41% of iatrogenic intradural aneurysm had ruptured within 2 months after the causative procedure [6].

Trapping or occlusion of the proximal parent artery with or without bypass surgery has been the procedure of choice for most posttraumatic aneurysms. Endovascular treatment of posttraumatic intracranial aneurysm has recently been described [2-5,8-10]. Simple parent artery occlusion with or without carotid test occlusion has been the preferred endovascular treatment in some studies [2,8,9]. In more recent articles, the authors have described endosaccular GDC embolization of the aneurysms and preservation of the parent vessel [3-5,10].

Of these, only few reports have dealt with the endosaccular coil embolization of intradural traumatic aneurysms [3,5,10]. Lempert et al treated 11 patients with pseudoaneurysms, of which only two were intradural lesions of the anterior cerebral artery.

They could embolize only one of them with preservation of the parent artery [3]. Lenthall et al treated a basilary tip pseudoaneurysm caused by endoscopic ventriculostomy with preservation of the parent artery in a child [10]. Tokunaga et al embolized successfully two intradural aneurysms: one developed after inadvertent arterial laceration the other after arterial injury during open surgery [5]. As far as we know, our patient is the fifth case in the literature having posttraumatic intradural aneurysm

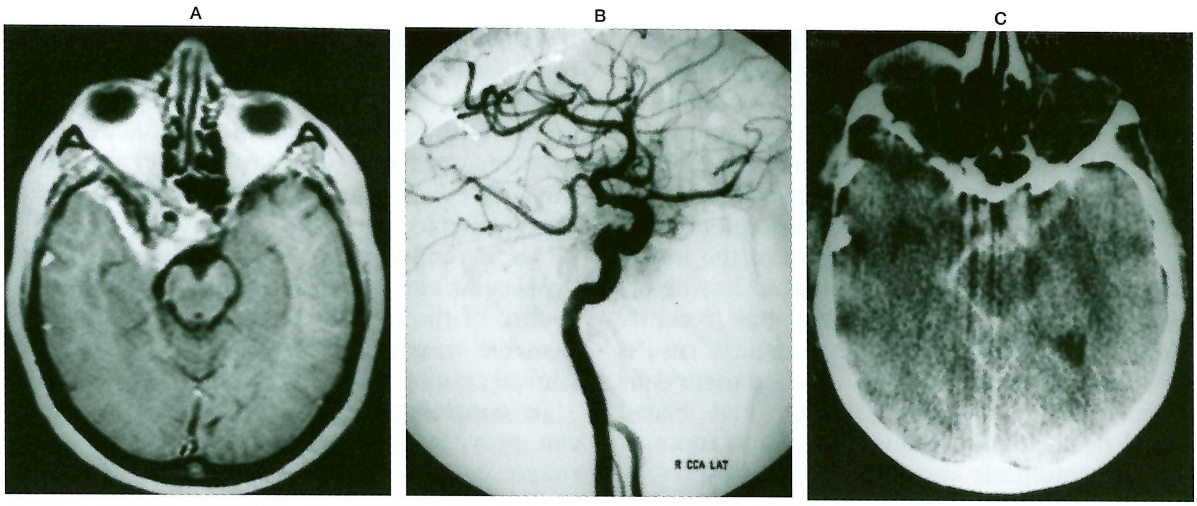
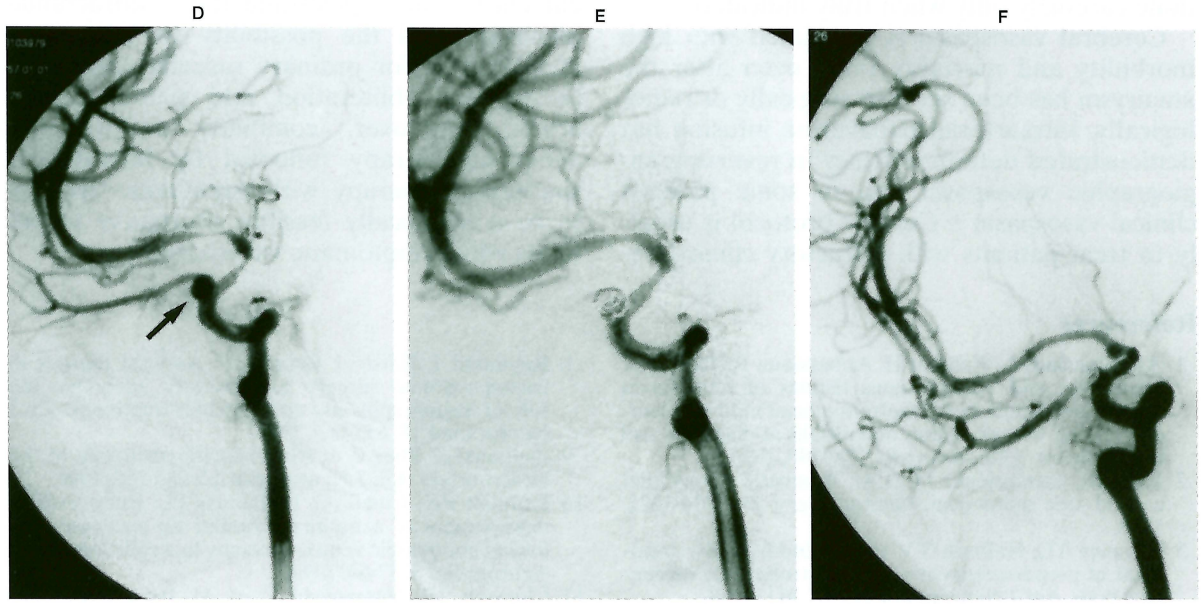


Figure 1 A) Preoperative contrast-enhanced T1-weighted axial MR image shows right sphenoid wing meningioma encasing the distal carotid artery. B) Preoperative right carotid angiogram, lateral view, shows faint tumoral opacification around the distal carotid artery, but no aneurysms. C) CT scan 15 days after surgery shows SAH prominent in basal cisterns. Note successful removal of the tumor under right-sided craniotomy defects. D) Right internai carotid angiogram, left anterior oblique view, 3 days after SAH shows a newly developed 5-mm saccular carotid aneurysm at the ophthalmic artery origin (arrow). Note severe vasospasm of the M1 segment and the distal internal carotid artery. E): Right internal carotid angiogram, left anterior oblique view, immediately after coil embolization and subsequent intraarterial papaverine infusion, shows complete obliteration of the aneurysm. Marked increase in calibre of the M1 segment and the distal carotid artery is evident. F) Right internal carotid angiogram, left anterior oblique view, 3 months after embolization, shows continuous total obliteration of the aneurysm.



treated successfully by GDC embolization and preservation of the parent artery.

Timing and indication of endosaccular embolization in case of the posttraumatic intracranial aneurysm is challenging and needs to be explained more clearly. According to Lempert et al, treatment of the pseudoaneurysm itself in the acute phase with preservation of the parent vessel may be fraught with complications owing to the fragility of the pseudowall of the pseudoaneurysm [3]. They have offered endosaccular embolization in the subacute phase to take advantage of the natural pathophysiology of the developing pseudoaneurysm, in which continuos fibrotic encapsulation strengthens and stabilizes its wall. Pseudoaneurysms occurring within the bony confines such as cavernous or petrous carotid, however, may provide indi-

cation for acute treatment by GDC embolization due to the fact that bony structures stabilize the aneurysm wall. Waiting for subacute period as suggested by Lempert et al may not be always possible. Some patients have to be treated as soon as possible to lower the risk of rebleeding, or to overcome consequences of SAH such as vasospasm. In spite of the inherent risks, we managed our patient as having ordinary saccular aneurysm that has recently bled, and obtained good radiological results without any complications. Pseudoaneurysms have usually complex structure and broad based neck. Unlike psaudoaneurysm, some of the posttraumatic intracranial aneurysm may have relatively small ostia [5] as in our patient. In this situation, the aneurysm may be successfully embolized with GDCs with preservation of the parent artery. Indeed, the decision of timing and indication for coil embolization of posttraumatic intracranial aneurysm must be individualized. One must keep in mind that coil embolization of such aneurysms should be done carefuJly only when truly indicated [5].

Cerebral vasospasm is associated with high morbidity and mortality rates, even after the aneurysm has been secured surgically or radiologically. Intraarterial papaverine infusion has demonstrated definite efficacy in reversing angiographic vasospasm and in some patients clinical vasospasm [11]. Current protocol is usually to treat patients with refractory clinical vasospasm at the earliest opportunity. In case of ruptured intradural aneurysm and symptomatic vasospasm, the aneurysm may be occluded with GDCs followed by intraarterial infusion of papaverine to treat vasospasm as an one-stage procedure [12]. Because of the risk of aneurysm rupture papaverine should be infused only distal to the aneurysm or after completion of endosaccular coil embolization. In one-stage procedure of the present patient, we obtained acceptable immediate angiographic and good late clinical results in the follow-up period.

In conclusion, although surgical treatment can prevent catastrophic intracranial hemorrhage, posttraumatic or iatrogenic aneurysms are usually difficult to eliminate without sacrificing the parent artery. Recent advances in endovascular techniques, however, allow safe approaches to complicated intracranial vascular lesions. Review of the literature dealing with endovascular treatment of posttraumatic intracranial aneurysms together with our experience simply demonstrated rather common occurrence of posttraumatic intracranial aneurysm and the possibility to treat such aneurysm as for ordinary saccular aneurysm: aneurysmal obliteration and patent parent artery. Moreover, combined endovascular aneurysm therapy followed by intraarterial spasmolytic therapy with papaverine appears to be a technically feasible alternative in patients with symptomatic vasospasm.

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